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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/529,762	04/18/2000	CHARLES W RITTERSHAUS	TCS-420.1PUS	3426

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EXAMINER
HUYNH, PHUONG N

ART UNIT	PAPER NUMBER
1644	

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Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/529,762	RITTERSHAUS ET AL.	
	Examiner	Art Unit	
	" Neon" Phuong Huynh	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *Three* MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *7/10/01; 10/22/01* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *38-52* is/are pending in the application.

4a) Of the above claim(s) *38,39,49 and 50* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *40-48, 51 and 52* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) *5* .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

DETAILED ACTION

1. Claims 38-52 are pending.

2. Applicant's election with traverse of Group III, Claims 40-48, 51 and 52, drawn to a method of modulating the level of endogenous active cholesteryl ester transfer protein (CETP) in a mammal, filed 10/22/01, is acknowledged. The traversal is on the grounds that Ha et al reference cited by examiner teaches administered human CETP to rats to increase plasma CETP activity, which resulted in a decreased level of HDL-cholesterol and an accompanying increased level of LDL-cholesterol while Applicants' invention is directed to administered a non-endogenous CETP to a mammal to cause an immune response against the mammal's own, endogenous CETP which in turn decreases (endogenous) CETP activity, increases the level of HDL-cholesterol, and decreases the level of LDL-cholesterol to unexpected levels. This is not found persuasive because of the reasons set forth in the restriction mailed 9/21/01. Further, the claims recite a method of **modulating** the level of endogeneous CETP in a mammal comprising administering to the mammal a whole, non-endogenous CETP in an amount effective to reduce CETP activity below that of the untreated mammal. The word "modulating" as recited in the claims can be increase or decrease in CETP activity. Finally, the claims as written do not indicate (1) administering a non-endogenous CETP to a mammal to **cause an immune response against the mammal's own, endogenous CETP** which in turn decreases (endogenous) CETP activity, increases the level of HDL-cholesterol, and decreases the level of LDL-cholesterol to unexpected levels and (2) the non-endogenous CETP is **humanized rabbit CETP molecules of SEQ ID NOS: 5 and 6**. As applicant point out in the traversal of restriction, Ha *et al* teach administering a non-endogenous CETP (human CETP) to a rat, which is a mammal, to modulate CETP activity and compared CETP activity to that of the untreated control. Upon reconsideration and in response to applicant's election with traverse to the restriction requirement separating Group IV, drawn to a method of modulating the level of endogenous CETP using allelic variant of the mammal's endogenous CETP and Group V, drawn to a method of modulating the level of endogenous CETP using non-endogenous CETP that has one or more amino acids deletion or substitution, is hereby withdrawn. Therefore, the requirement of Group III (now claims 40-48, 51 and 52) drawn to a method of modulating the level of endogenous active CETP comprising administering to the mammal a whole, non-endogenous CETP from xenogeneic CETP, allelic

variant, and modified CETP having one or more amino acid deletion or substitution is still deemed proper and is therefore made FINAL.

3. Claims 38-39 and 49-50 are withdrawn from further consideration by the examiner, 37 C.F.R. 1.142(b) as being drawn to non-elected inventions.

4. Claims 40-48, 51 and 52 are being acted upon in this Office Action.

5. Applicant should amend the first line of the specification to indicate that this application claims benefit of the provisional application 60/109,804, filed 10/20/1997.

6. Applicant should provide a certified copy for foreign priority claims made under the provisions of 35 U.S.C. 119(a)-(d) as stated in the declaration.

7. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

8. Claims 40-48 and 51-52 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling only for (1) a method of increasing the production of anti-CETP antibody wherein the method comprises administering to a rabbit a C-terminus of human CETP peptide conjugated to tetanus toxoid consisting of SEQ ID NO: 7 or a whole recombinant human CETP consisting of SEQ ID NO: 1 (See page 17 lines 2-3 of the specification) for reducing CETP activity, increasing the HDL-cholesterol, and lowering LDL-cholesterol associated with atherosclerosis, does not reasonably provide enablement for (1) a method of modulating the level of endogenous, active cholesteryl ester transfer protein (CETP) in *any* mammal comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to reduce CETP activity below 20% of that the untreated mammal, (2) a method of modulating the level of endogenous cholesteryl ester transfer protein (CETP) in a mammal comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to achieve a level of essentially 0 μg of CETP per milliliter of blood of the mammal, (3) a method of modulating the level of HDL-cholesterol in a mammal comprising administering to

the mammal *any* whole, non-endogenous CETP in an amount effective to achieve a lipoprotein profile wherein greater than about 90% of the total cholesterol in the blood of the mammal is HDL-cholesterol, (4) the method of modulating the level of HDL-cholesterol in a mammal comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to achieve a lipoprotein profile wherein greater than about 100% of the total cholesterol in the blood of the mammal is HDL-cholesterol, (5) a method of modulating the level of LDL-cholesterol in a mammal comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to achieve a lipoprotein profile wherein less than about 10% of the total cholesterol in the blood of the mammal is LDL-cholesterol, (6) a method of modulating the level of LDL-cholesterol in a mammal comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to achieve a lipoprotein profile wherein essentially none of the total cholesterol in the blood of the mammal is LDL-cholesterol, (7) the method wherein the mammal is a human, (8) the method wherein *any* whole, non-endogenous cholesteryl ester transfer protein is any xenogeneic CETP, *any* allelic variant of *any* mammalian's endogenous CETP, *any* mammalianized non-endogenous CETP in which the amino acid sequence of a non-endogenous CETP ahs been altered by deletion or substitution of one or more amino acids so as to make the amino acid sequence of said non-endogenous CETP more similar to the mammal's endogenous CETP, (9) any method mentioned above in combination with an adjuvant such as the ones recited in claim 52 for non-specifically stimulate the immune response of the mammal for a vaccine against LDL-cholesterol. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in **scope** with these claims.

Factors to be considered in determining whether undue experimentation is required to practice the claimed invention are summarized *In re Wands* (858 F2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)). The factors most relevant to this rejection are the scope of the claim, the amount of direction or guidance provided, the lack of sufficient working examples, the unpredictability in the art and the amount of experimentation required to enable one of skill in the art to practice the claimed invention. The specification disclosure is insufficient to enable one skilled in the art to practice the invention as broadly claimed without an undue amount of experimentation.

The specification discloses only a full-length rabbit CETP polypeptide consisting of SEQ ID NO: 3, a full-length human CETP polypeptide consisting of SEQ ID NO: 1, two

mammalianized rabbit CETP polypeptides consisting of SEQ ID NOS: 5 and 6 and a method of vaccinating a rabbit, which is a mammal, with a recombinant human CETP of SEQ ID NO: 1, or a peptide of the C-terminus of human CETP conjugated to tetanus toxoid of SEQ ID NO: 7 (See page 16-17, Figs 8-9 of the specification) for increasing the titer of CETP specific antibodies in the plasma, wherein said antibody cross-react with the rabbit CETP c-terminal peptide from amino acids 477-496 of SEQ ID NO: 3, thereby reducing the CETP activity below 20% of the untreated control at 14 weeks (Fig 9), reducing the level of native CETP to 0 μg/ml of blood of the mammal at 14 weeks post immunization, increasing the level of HDL-cholesterol to greater than 90% of the total cholesterol in the blood of the mammal, increasing the level of HDL-cholesterol to about 100% of the total cholesterol in the blood of the mammal 14 weeks after immunization (Fig 9), reducing the level of LDL-cholesterol of less than about 10% of the total cholesterol in the blood plasma of the mammal (See Fig 7). The specification further discloses on page 5 line 23 that non-endogenous CETP can be from another mammalian species such as rabbit CETP, mouse CETP or simian CETP for administration to a human.

Other than the specific polypeptides mentioned above for a method of inhibiting the endogenous CETP activity, the specification fails to provide any guidance as how to make and use *any* non-endogenous CETP for a method of modulating any endogenous CETP in any mammal. There is insufficient guidance and working examples as to which amino acid residues within any of the non-endogenous CETP of any mammal can be deleted, substitute and whether the resulting modified CETP protein would maintain the structure and function as SEQ ID NO: 7 and 1, in turn, generating antibodies that would bind specifically to the human or the rabbit CETP for a method of inhibiting any endogenous CETP activity associated with atherosclerosis. Given the indefinite number of undisclosed non-endogenous CETP protein, it is unpredictable which undisclosed non-endogenous CETP would be useful for a method of inhibiting any non-endogenous CETP activity associated with atherosclerosis.

Ngo *et al* teach that the amino acid positions within the polypeptide/protein that can tolerate change such as conservative substitution or no substitution, addition or deletion which are critical to maintain the protein's structure/function will require guidance (see Ngo et al., 1994, The Protein Folding Problem and Tertiary Structure Prediction, pp. 492-495).

It is well known in the art at the time the invention was made that antibody epitopes (B cell epitopes) are not linear and are comprised of complex three-dimentional array of scattered residues which will fold into specific conformation that contribute to binding (See Kuby 1994,

page 94, in particular). Immunization with a peptide comprising a contiguous amino acid sequence of 8 amino acid residues or a protein derived from a full-length polypeptide may result in **antibody specificity** that differs from antibody specificity directed against the native full-length polypeptide. Without the specific amino acid residues, it is unpredictable to determine which antibody response generated from any CETP polypeptide and fragment thereof will have the same antibody specificity as an antibody generated from the SEQ ID NO: 1 and 7, in turn, can be use for a method of reducing CETP activity associated with atherosclerosis.

Furthermore, Marrott *et al* (PTO 1449) teach that transgenic mice expressing exogenous simian cholesteryl ester transfer protein (CETP) results in severe atherosclerosis with a marked increases in the concentration of LDL-cholesterol and a decrease in the concentration of HDL-cholesterol (See entire document, Abstract, in particular). Likewise, Breslow *et al* (PTO 1449) teach human CETP transgenic mice overexpressing human CETP has lower level of HDL-cholesterol the effects CETP were less than expected based on studies comparing normal and CETP-deficient humans (See page 8316, column 2, CETP transgenic mice, in particular). These results indicate that not all xenogeneic none-endogenous CETP are appropriate for a method of modulating CETP activity, particularly in humans, where atherosclerosis is multi-factorial complex disease.

With regard to allelic variant of *any* mammal's endogenous CETP, the specification defines allelic variant is a polymorphism of human CETP producing by another human individual (See page 8, lines 20 of the specification). However, the specification discloses only **one** human CETP polypeptide consisting of SEQ ID NO: 1. There are no additional human CETP which have been demonstrate to be useful for immunizing any mammal, in turn, for a method of modulating any endogenous CETP within any mammal. Given the indefinite number of undisclosed non-endogenous CETP protein, it is unpredictable which undisclosed non-endogenous CETP would be useful for a method of inhibiting any non-endogenous CETP activity associated with atherosclerosis. It follows that any xenogeneic CETP, any allelic variant of any mammalian's endogenous CETP, any mammalianized non-endogenous CETP other than SEQ ID NO: 5 and 6 are not enable.

Regarding claims 46 and 48 wherein the mammal is a human, there are no working examples in the specification as filed to demonstrate that administering any whole non-endogenous CETP such as recombinant human CETP is effective in reducing CETP activity below 20% of that untreated human, reducing the level of essentially 0 μg of CETP per ml of

blood of the human, increasing the level of HDL-cholesterol to greater than about 90% or about 100% of the total cholesterol in the blood of the human, lowering the level of LDL-cholesterol to less than 10% of the total cholesterol in the blood plasma or reducing the level of LDL-cholesterol to essentially none as recited in claim 45. Furthermore, since CETP is a "self" protein, it is not clear in the specification as filed how administering a whole recombinant human CETP (which is not foreign and no different than one's own CETP) would induce endogenous **antibody** direct toward one's own CETP at a level **sufficient high** to modulate one's own level of endogenous CETP activity. Stevens *et al* teach in order induce high levels of antibodies reactive to one's own protein (break immune tolerance) such as hCG for a contraceptive vaccine, the protein must be conjugated to a foreign protein or carrier molecule such as KLH or tetanus toxoid to enhance the immunogenicity of the hCG protein. In re wands, 858 F.2d at 737, 8 USPQ2d at 1404 (Fed. Cir. 1988), the decision of the court indicates that the more unpredictable the area is, the more specific enablement is necessary. In view of the quantity of experimentation necessary, the limited working examples, the unpredictability of the art, the lack of sufficient guidance in the specification and the breadth of the claims, it would take an undue amount of experimentation for one skilled in the art to practice the claimed invention.

9. Claims 40-48 and 51-52 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor, at the time the application was filed, had possession of the claimed invention.

The specification does not reasonably provide a **written description** of (1) a method of modulating the level of endogenous, active cholesteryl ester transfer protein (CETP) in *any* mammal comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to reduce CETP activity below 20% of that the untreated mammal, (2) a method of modulating the level of endogenous cholesteryl ester transfer protein (CETP) in a mammal comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to achieve a level of essentially 0 μg of CETP per milliliter of blood of the mammal, (3) a method of modulating the level of HDL-cholesterol in a mammal comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to achieve a lipoprotein profile wherein greater than about 90% of the total cholesterol in the blood of the mammal is HDL-cholesterol, (4) the method of modulating the level of HDL-cholesterol in a mammal

comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to achieve a lipoprotein profile wherein greater than about 100% of the total cholesterol in the blood of the mammal is HDL-cholesterol, (5) a method of modulating the level of LDL-cholesterol in a mammal comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to achieve a lipoprotein profile wherein less than about 10% of the total cholesterol in the blood of the mammal is LDL-cholesterol, (6) a method of modulating the level of LDL-cholesterol in a mammal comprising administering to the mammal *any* whole, non-endogenous CETP in an amount effective to achieve a lipoprotein profile wherein essentially none of the total cholesterol in the blood of the mammal is LDL-cholesterol, (7) the method wherein the mammal is a human, (8) the method wherein *any* whole, non-endogenous cholesteryl ester transfer protein is any xenogeneic CETP, *any* allelic variant of *any* mammalian's endogenous CETP, *any* mammalianized non-endogenous CETP in which the amino acid sequence of a non-endogenous CETP ahs been altered by deletion or substitution of one or more amino acids so as to make the amino acid sequence of said non-endogenous CETP more similar to the mammal's endogenous CETP, (9) any method mentioned above in combination with an adjuvant such as the ones recited in claim 52 for non-specifically stimulate the immune response of the mammal for a vaccine against LDL-cholesterol.

The specification discloses only a full-length rabbit CETP polypeptide consisting of SEQ ID NO: 3, a full-length human CETP polypeptide consisting of SEQ ID NO: 1, a mammalianized rabbit CETP consisting of SEQ ID NOS: 5 and 6 and a method of vaccinating a rabbit, which is a mammal, with a recombinant human CETP of SEQ ID NO: 1, or a peptide of the C-terminus of human CETP conjugated to tetanus toxoid of SEQ ID NO: 7 (See page 16-17, Figs 8-9 of the specification) for increasing the titer of CETP specific antibodies in the plasma, wherein said antibody cross-react with the rabbit CETP c-terminal peptide from amino acids 477-496 of SEQ ID NO: 3, thereby reducing the CETP activity below 20% of the untreated control at 14 weeks (Fig 9), reducing the level of native CETP to 0 μg/ml of blood of the mammal at 14 weeks post immunization, increasing the level of HDL-cholesterol to greater than 90% of the total cholesterol in the blood of the mammal, increasing the level of HDL-cholesterol to about 100% of the total cholesterol in the blood of the mammal 14 weeks after immunization (Fig 9), reducing the level of LDL-cholesterol of less than about 10% of the total cholesterol in the blood plasma of the mammal (See Fig 7).

With the exception of the specific CETP polypeptides mentioned above, there is insufficient written description about the structure associated with functions of *any* non-endogenous CETP wherein said endogenous CETP is *any* xenogeneic CETP, *any* allelic variant of any mammalian's endogenous CETP, *any* mammalianized non-endogenous CETP having one more amino acid altered by deletion, or substitution as to make the amino acid sequence more similar to the mammal's endogenous CETP for a method of modulating the level of endogenous active cholesteryl ester transfer protein associated with atherosclerosis.

Given the lack of a written description of *any* additional representative species of allelic variant of *any* human CETP such as any naturally occurring polymorphism as encompassed by the claims, one of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species to describe the genus. Thus, Applicant was not in possession of the claimed genus. *See University of California v. Eli Lilly and Co. 43 USPQ2d 1398.*

Applicant is directed to the Revised Interim Guidelines for the Examination of Patent Applications Under the 35 U.S.C. 112, 1 "Written Description" Requirement, Federal Register, Vol. 66, No. 4, pages 1099-1111, Friday January 5, 2001.

10. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office Action:

A person shall be entitled to a patent unless –

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

11. Claims 40-44, 45, 47 and 51-52 are rejected under 35 U.S.C. 102(a) as being anticipated by the WO 96/39168 publication (Dec 12, 1996, PTO 892).

The 96/39168 publication teaches a method of modulating the endogenous active cholesteryl ester transfer protein (CETP) in a mammal such as a rabbit comprising administering to said mammal a full-length human CETP of SEQ ID NO: 1 of WO 96/39168, or a taxoid conjugated human CETP peptide, which are non-endogenous CETP, in an amount effective to stimulate an immune response such as anti-CETP antibody wherein said antibody inhibits the function of CETP such as reducing the CETP activity below 20% of that of the untreated mammal (See abstract, Fig 2, of WO 96/39168, in particular). The reference method comprises administered to the mammal in combination with an adjuvant such as CFA (Complete Freund's Adjuvant) or IFA (Incomplete Freund's adjuvant) wherein the reference adjuvant is effective to non-specifically stimulate the immune response of the mammal such as production of antibody

(See page 7, line 29, page 8, lines 1-2, in particular). The reference method decreases LDL-cholesterol to less than 16% of the total cholesterol in the serum (blood plasma), which is about 10% (See Table 1, page 11, in particular). The term "about" expands the claimed 10% of the total cholesterol to read on the reference 16%. Claim 47 is included in this rejection because the reference teaches xenogeneic CETP which is a human CETP, in addition to a mammalianized non-endogenous CETP (See SEQ ID NO: 3 of WO 96/39168) where the reference SEQ ID NO: 3 is common to both human and rabbit CETP, which makes the human CETP more similar to rabbit and vice versa and the epitope is recognized by anti-CETP monoclonal antibody to which it is neutralized (See page 7, lines 20-22, in particular).

While the reference is silent that the reference method of administering to the mammal a whole non-endogenous CETP has the property of that recited in claims 41-43 and 45, the antibody directed against said non-endogenous CETP in the mammal and the functional properties of the reference antibody are the inherent property of the reference method. Therefore the claimed method appears to be the same as the prior art method. Since the Patent Office does not have the facilities for examining and comparing the method of the instant invention to those of the prior art, the burden is on applicant to show that the prior art method is different from the claimed method. See In re Best, 562 F.2d 1252, 195 USPQ 430(CCPA 1977). Thus, the reference teachings anticipate the claimed invention.

12. No claim is allowed.

13. Any inquiry concerning this communication or earlier communications from the examiner should be directed to "Neon" Phuong Huynh whose telephone number is (703) 308-4844. The examiner can normally be reached Monday through Friday from 9:00 am to 6:00 p.m. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christina Chan can be reached on (703) 308-3973. Any inquiry of a general nature or relating to the status of this application should be directed to the Technology Center 1600 receptionist whose telephone number is (703) 308-0196.

14. Papers related to this application may be submitted to Technology Center 1600 by facsimile transmission. Papers should be faxed to Technology Center 1600 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform to the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center telephone number is (703) 305-7401.

Phuong N. Huynh, Ph.D.
Patent Examiner
Technology Center 1600
January 28, 2002

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